IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 6 Ser. 11 (June. 2019), PP 39-42
www.iosrjournals.org

Primary Aldosteronism Due To Unilateral Adrenal Adenoma: A Case Report.

Ohenhen Oluwatoyin.A[1,2], Okocha Kingsley. E[1]
[1]*Endocrine Unit, Department Of Medicine, University Of Benin Teaching Hospital, Benin City, Edo State Nigeria.*
[2]*Corresponding author: Dr. Oluwatoyin .A. OHENHEN*
Department of Medicine University of Benin Teaching Hospital.

Abstract
BACKGROUND*: Primary aldosteronism as a cause of secondary hypertension is frequently overlooked and underdiagnosed. This case report highlights the importance of screening for aldosteronism in patients with hypertension and hypokalemia.*
METHOD*: The case record of the index patient.*
RESULT*: A 41year old female civil servant, referred to the endocrine clinic from a peripheral center on account of persistent hypokalemia. Hypokalemia was diagnosed accidentally during a routine medical screening 5 years prior to presenting at the endocrine clinic. There was positive history of polyuria and nocturia. She was also diagnosed hypertensive at about the same time and commenced on Amlodipine. General examination and systemic examination were essentially normal, with pulse rate of 68 beats per minute and blood pressure of 130/70mmHg. Laboratory investigations revealed serum potassium of 1.5 mmol/L, Urinary Potassium Excretion of 58 mmol/24hours (25–126 mmol/24 hours), Plasma Aldosterone Concentration was markedly elevated : 1370 pmol/L (11.7-236.0). Plasma Renin level was very low, less than 0.3ng/L (1.7-236.0). Computed Tomography scan of the abdomen revealed a "left supra renal mass, Probably Adrenal cortical adenoma". A diagnosis of Conn's Syndrome was made and she was commenced on an aldosterone antagonist (spironolactone), and anti-hypertensives. Weekly monitoring of serum potassium showed a gradual rise, with serum potassium being normalized by the sixth week of treatment. She was subsequently referred to the Urology unit of the hospital where she is currently being evaluated for adrenalectomy.*
CONCLUSION*: Primary Aldosteronism should be considered in patients presenting with hypertension and persistent hypokalemia who are not on diuretics.*
Key Words*; Hypokalemia, Hypertension, and Primary Aldosteronism*

Date of Submission: 02-06-2019 Date of acceptance: 17-06-2019

I. Introduction:

Primary Aldosteronism (PA) is the commonest cause of secondary arterial hypertension[1]. Litynski[2] reported the first cases, however, Conn, an American Physician was the first to well characterize the disorder, in 1955. Conn syndrome, as originally described, refers specifically to primary aldosteronism due to the presence of an adrenal aldosteronoma (aldosterone-secreting benign adrenal neoplasm)[1]. The cardinal anomalies causing primary aldosteronism syndrome is autonomous (non-suppressible) aldosterone production, suppressed and poorly stimulative levels of plasma renin, coexisting with only mildly expanded intravascular and extravascular fluid volume. Other etiologies of primary hyper-secretion of aldosterone include bilateral adrenal hyperplasia (BAH), glucocorticoid-remediable aldosteronism and very rare Adrenocortical carcinoma[3,4].

Primary hyper-aldosteronism as a cause of secondary hypertension is frequently overlooked and underdiagnosed despite the fact that it accounts for 5-15% of Secondary Hypertension[1]. This case report highlights the importance of screening for hyperaldosteronism in patients with hypertension and spontaneous hypokalemia.

II. Case Presentation

A 41year old female civil servant, referred to the endocrine clinic from a peripheral center on account of persistent hypokalemia. Hypokalemia was diagnosed incidentally during a routine medical screening 5 year prior to presentation at which time also, she was diagnosed hypertensive and commenced on Amlodipine. There was positive history of polyuria and nocturia. There was however no history of headaches, palpitation, exertional dyspnea, orthpnoea, leg or facial swelling. Physical examination revealed a calm middle aged woman who was in no obvious distress and had no pedal edema. Cardiovascular examination revealed a pulse rate of 68

beats per minute, Blood pressure of 130/70mmHg, and the Jugular Venous pressure was not elevated. She had a displaced apex beat and first and second heart sounds only were heard. Other physical examinations were unremarkable.

Laboratory investigations revealed electrolyte with serum potassium of 1.5 mmol/L, sodium of 131 mmol/L, bicarbonate of 19mmol/L, chloride of 96mmol/L. The results of subsequent potassium profile is shown in Table 1

TABLE1. Serial Serum Potassium (mmol/L)

24-10-17	30-11-17	10-12-17	21-12-17	29-01-18
1.8	2.0	1.9	2.2	2.5

Other investigations include; Urinary Potassium Excretion of 58 mmol/24hours (25–126 mmol/24 hours), Plasma Aldosterone Concentration was markedly elevated:1370 pmol/L (11.7-236.0). Plasma Renin level was very low, less than 0.3ng/L (1.7-23.9). The 24hrs Urinary potassium was 58mmol/L (Reference: 25-125mmol/L) {Urine volume: 2220mls (600-2400)}

Computed Tomography scan of the abdomen revealed a "left supra renal mass, Probably Adrenal cortical adenoma". A diagnosis of Conn's Syndrome was made and she was commenced on an aldosterone antagonist (spironolactone), and Amlodipine.

Weekly monitoring of serum potassium showed a gradual rise, with serum potassium being normalized by the sixth week of treatment as shown in Table 2

TABLE 2 SERIAL SERUM POTASSIUM RESULT POST TREATMENT (mmol/L)

06-02-18	20-02-18	01-03-18	08-03-18	15-03-18	22-03-18
2.9	3.1	3.3	3.8	4.3	4.5

She was subsequently referred to the Urology unit of the hospital where she is currently being evaluated for adrenalectomy.

III. Discussion

This case highlights the importance of adequate investigation in the management of a hypertensive patient. The finding of spontaneous hypokalemia in a known hypertensive prompted further investigations which led eventually to the diagnosis of a Primary aldosteronism secondary to an Adrenal Adenoma.

Conn's syndrome[6], which refers specifically to Primary aldosteronism due to the presence of an aldosteronoma (aldosterone-secreting benign adrenal neoplasm), is characterized by increased aldosterone production, suppressed plasma renin activity, hypertension, hypokalemia and metabolic alkalosis. Although hyperkalaemia is a prominent feature of Primary aldosteronism, normokalemia does not exclude the diagnosis of primary hyperaldosteronism[1, 3].

Primary aldosteronism may occur in as many as 5-15% of Hypertensive patients and this is estimated to be higher in patients with resistant hypertension[1]. Renata et al[7] reported that primary aldosteronism was diagnosed in 16.6% (58) of 350 hypertensive patients they studied, while hypokalaemia occurred in 25 patients (43.1%) of persons with primary aldosteronism. A diagnosis of Primary aldosteronism is made with biochemical and imaging studies. The Endocrine Society guidelines on PA[8] recommend screening high-risk groups for PA. These high risk groups include: (i) patients with Hypertension of Joint National Commission stage 2 (>160–179/100–109 mm Hg), stage 3 (>180/110 mm Hg)with sustained blood pressure on each of three measurements obtained on different days; (ii) patients with hypertension resistant to three conventional antihypertensive drugs (including a diuretic) or controlled blood pressure on four or more antihypertensive drugs; (iii) patients with hypertension and spontaneous or diuretic-induced hypokalaemia; (iv) patients with hypertension and adrenal incidentaloma; (v) patients with hypertension and sleep apnoea; (vi) patients with hypertension and a family history of early onset hypertension or cerebrovascular accident at a young age (<40 years); and (vii) all hypertensive first-degree relatives of patients with PA

Screening tests for primary aldosteronism include serum potassium and bicarbonate levels, Plasma renin activity (PRA) or plasma renin concentration (PRC) and Plasma aldosterone concentration (PAC)/plasma renin activity ratio[1]. In patients with primary aldosteronism, Plasma aldosterone concentration is usually elevated while PRC is suppressed whereas, in secondary hyperaldosteronism (e.g renovascular hypertension) should be considered if both PAC and PRA are elevated[3] and the PAC/PRA ratio is <277 (with PAC measured in pmol L^{-1} and PRA in ng mL^{-1} h^{-1}; PAC/PRA ratio <10 if PAC is measured in ng dL^{-1} and PRA in ng mL^{-1} h^{-1}). A PAC >277 pmol L^{-1} (>10 ng dL^{-1}) and a PRA <1.0 ng mL^{-1} h^{-1} or a PRC lower than the lower limit of the reference range is a positive case detection test result, a finding that warrants further testing[8]

A high PAC and low PRA test result is not diagnostic of PA and thus must be followed up by confirmatory tests which demonstrates inappropriate or autonomous aldosterone secretion[8,9]. The confirmatory tests is however not necessary and need not be performed in the setting of spontaneous hypokalaemia with PAC >555 pmol L^{-1} (>20 ng dL^{-1}) and PRA <1 ng mL^{-1} h^{-1} (or PRC below the lower limit of the reference range); this presentation is diagnostic of PA[8,10](as in this index case, thus confirmatory tests were not done) All other patients should have PA confirmed by demonstration of aldosterone secretory autonomy with aldosterone-suppression testing, which can be performed with orally administered sodium chloride and measurement of urinary aldosterone excretion or with intravenous sodium chloride loading and measurement of PAC[3,10,11]. These confirmatory test include oral saline loading test, Intravenous saline infusion test, and the now less commonly used fludrocortisone suppression and captopril stimulation tests.[8]

All patients with PA should undergo adrenal CT as the initial study in subtype testing. CT scan has a sensitivity of 67-85% in patients with primary hyperaldosteronism[12].

Magnetic resonance imaging has no advantage over CT in subtype evaluation of PA[1] Adrenal venous sampling following cosyntropin stimulation is used in patients with equivocal CT scan findings. This method remains the gold standard test to distinguish unilateral from bilateral disease in patients with PA[13,14]. Adrenal venous sampling is a technically demanding procedure because the right adrenal vein is small and may be difficult to locate and cannulate; the success rate depends on the expertise and engagement of the interventional radiologist[15,16]

The goal of subtype testing is to determine whether the source of aldosterone excess is from the right, left or both adrenal glands. When localized to one adrenal gland (Aldosterone producing adenoma or Primary Adrenal Hyperplasia), unilateral adrenalectomy results in normalization of hypokalaemia in all patients; hypertension is improved in all patients and is cured in 30–69%[1,3,17,18]. Unilateral laparoscopic adrenalectomy is the surgical procedure of choice in patients Conn's syndrome, and its long term cure rate averages 69%.[18]. In patients with BAH, unilateral adrenalectomy does not cure the aldosterone excess thus medical treatment with mineralocorticoid receptor antagonists (Spironolactone or Eplerenone) is the mainstay of treatment. Hypokalemia tends to correct with adequate doses of Spironolactone, so potassium supplementation may not be required.

Among the major goals of therapy for primary aldosteronism are ; normalization of blood pressure as well as prevention of the morbidity and mortality associated with hypertension, normalization of serum potassium and other electrolytes, and normalization of serum aldosterone. The general guidance that should be provided to all patients with hypertension is also appropriate for patients with PA: maintain ideal body weight, avoid tobacco, participate in a regular exercise programme and follow a sodium-restricted diet.

IV. Conclusion

PA is a relatively common cause of hypertension and can be either managed with surgery or specifically targeted with pharmacologic therapy. Undetected or ineffectively treated PA results in increased cardiovascular morbidity and nephrotoxicity. Proper investigation of the hypertensive patient is recommended especially those with hypokalaemia. This case highlights the importance of a high index of suspicion and proper investigation in cases of hypertension with spontaneous hypokalemia.

References

[1]. Uwaifo GI, Sarlis NJ, Khardori R. Primary Aldosteronism. March 02, 2018. " Retrived from": https://emedicine.medscape.com/article/127080.
[2]. Kucharz EJ . Michał Lityński—a forgotten author of the first description on primary hyperaldosteronism. Pol Arch Med Wewn 2007;117:57–58
[3]. Young WF. Diagnosis and treatment of primary aldosteronism: practical clinical perspectives. Journal of Internal Medicine 2019; 285:12-148.
[4]. DeGroot L, Jameson J. Endocrinology. 4th edn. Philadelphia: W.B. Sanders, 2001.
[5]. Melby JC. Diagnosis and treatment of primary aldosteronism and isolated hypoaldosteronism. Clin Endocrinol Metab 1985;14:977–95
[6]. Conn JW. Presidential address. I. Painting background. II. Primary aldosteronism, a new clinical syndrome. J Lab Clin Med 1955; 45: 3–17.
[7]. Renata M, Agnieszka J, Wojciech Z. The prevalence of primary aldosteronism (PA) in a group of 350 hypertensive patients. Arterial Hypertension 2015; 19:9–12.
[8]. Funder JW, Carey RM, Mantero F et al. The management of primary aldosteronism: case detection, diagnosis, and treatment: an endocrine society clinical practice guideline. J Clin Endocrinol Metab 2016; 101: 1889–916.
[9]. Vakkalanka S, Zhao A, Samannodi M. Primary hyperaldosteronism: a case of unilateral adrenal hyperplasia with contralateral incidentaloma. Case Reports. 2016; 2016: bcr2016216209
[10]. Young WF. Primary aldosteronism: renaissance of a syndrome. Clin Endocrinol (Oxf) 2007; 66: 607–18.
[11]. Funder JW, Carey RM, Fardella C et al. Case detection, diagnosis, and treatment of patients with primary aldosteronism: an endocrine society clinical practice guideline. J Clin Endocrinol Metab 2008; 93: 3266–81.
[12]. Geisinger MA, Zelch MG, Braro E.L. Primary hyperaldosteronism: diagnosis, comparison of CT, adrenal venography and venous sampling. AM J Radiol 1983;141:299302.
[13]. Stowasser M. Update in primary aldosteronism. J Clin Endocrinol Metab 2015; 100: 1–10.

[14]. Young WF, Stanson AW, Thompson GB, Grant CS, Farley DR, van Heerden JA. Role for adrenal venous sampling in primary aldosteronism. Surgery 2004; 136: 1227–1235.
[15]. Dekkers T, Prejbisz A, Kool LJ et al. Adrenal vein sampling versus CT scan to determine treatment in primary aldosteronism: an outcome-based randomised diagnostic trial. Lancet Diabetes Endocrinol 2016; 4: 739–746.
[16]. Young WF, Stanson AW. What are the keys to successful adrenal venous sampling (AVS) in patients with primary aldosteronism? Clin Endocrinol (Oxf) 2009; 70: 14–17
[17]. Lim V, Guo Q, Grant CS et al. Accuracy of adrenal imaging and adrenal venous sampling in predicting surgical cure of primary aldosteronism. J Clin Endocrinol Metab 2014; 99: 2712–9
[18]. Sawka AM, Young WF, Thompson GB, Grant CS, Farley DR, Leibson C, van Heerden JA. Primary aldosteronism: factors associated with normalization of blood pressure after surgery. Ann Intern Med. 2001;135:258–261.

Dr. Oluwatoyin .A. "Primary Aldosteronism Due To Unilateral Adrenal Adenoma: A Case Report." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 6, 2019, pp 39-42.